ENVIRONMENTAL TOBACCO SMOKE IS NOT A PROVEN OCCUPATIONAL HAZARD

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TO THE

MARYLAND OCCUPATIONAL SAFETY AND HEALTH ADVISORY BOARD

DECEMBER 9 , 1993

SUMMARY

* ETS IS NOT EQUIVALENT TO THE SMOKE INHALED BY SMOKERS. ALTHOUGH GENERALLY BEYOND MATERIAL DETECTION, POSTULATED ETS COMPONENTS WOULD BE ORDERS OF MAGNITUDE BELOW LEVELS OFFICIALLY PERMITTED IN WORKPLACES.

* EPA'S CLAIM THAT ETS IS A LUNG CANCER RISK STEMS FROM THE SELECTIVE AND IMPROPER USE OF EPIDEMIOLOGIC DATA. EPIDEMIOLOGIC REPORTS OF WORKPLACE ETS EXPOSURE DO NOT SUPPORT CLAIMS OF INCREASED LUNG CANCER RISK.

* EPIDEMIOLOGIC STUDIES DO NOT SUGGEST THAT ETS IS A CARDIOVASCULAR RISK FACTOR IN THE WORKPLACE.

* THE SURGEON GENERAL AND OTHERS REPORT NO STATISTICALLY SIGNIFICANT ASSOCIATION OF ETS AND RESPIRATORY DISEASES IN ADULTS.

* EPA'S REPORT ON ETS IS NOT SCIENTIFICALLY JUSTIFIED. IT IS A POLITICAL GAMBIT IN A LONG EPA TRADITION OF BENDING SCIENCE TO SUIT POLICY AIMS.

My name is Gio Gori. I am a toxicologist with training in epidemiology and broad interests in smoking and health, cancer causation and risk assessment. I am President of the International Society of Regulatory Toxicology and Pharmacology, and Fellow of the Academy of Toxicological Sciences. In the 60's and 70's I was Deputy Director for Cancer Cause and Prevention at the National Cancer Institute, where I received the Public Health Superior Service Award in 1977 for separate activities as Director of the Smoking and Health Program. My resume is appended to this statement.

This statement is presented at the request of The Tobacco Institute. The views expressed, however, are my own and do not necessarily represent the position of The Tobacco Institute.

Recent interest in ETS has been motivated by the report on ETS released by the U.S. Environmental Protection Agency (EPA) on January 7, 1993 (1). Therefore, my comments will address the scientific merits of that report, as well as other data not addressed in the report and contrary to its conclusions.

I shall proceed according to standard scientific criteria of objective verification. Science is the mother of all technological innovations that sustain our civilization. In this role, science has achieved a deserved reputation for objectivity. Scientists seek objectivity by rules similar to the evidentiary rules observed in a court of justice. No effect is assumed until proven, while proof requires direct or indirect involvement, elimination of alibis, reliable witnesses, material evidence, and so on.

In this light, my assessment also rests on the premise that the Maryland Occupational Safety and Health Advisory board (MOSHAB) has an initial and paramount interest in determining how reliable a foundation of fact the Board is dealing with.

The EPA case rests on the claim that ETS is equivalent to the smoke that smokers inhale, and on the claim that epidemiologic studies of people who report exposure to ETS have a higher risk of lung cancer. The question is then: are these claims independently and objectively justifiable?

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The EPA itself is not sure that ETS is equivalent to the smoke that smokers inhale, and both asserts and denies this notion in different chapters of the ETS report (1. pages 2-9 and 6-6). Indeed, there are good reasons for its equivocations throughout the report.

ETS derives from the side-stream smoke produced by smoldering cigarettes, and from the small residues of main-stream smoke exhaled by active smokers. Generated and existing under much different conditions, these distinct smokes have some similarities but marked differences in chemical and physical composition and behavior.

Of the several thousand components identified in main-stream smoke, only a few hundred have been detected in side-stream smoke, due to greater dilutions. Because of even more extreme dilutions, fewer than 50 ETS components have been identified directly under field conditions, stretching the sensitivity limits of current analytical capabilities (2,3). Indeed, the compilers of reports from the National Academy of Sciences (4), the US Surgeon General (5) and the Environmental Protection Agency

(1,6,7) have been forced to infer the presence of ETS components by proxy, based on the composition of side-stream smoke from which ETS derives. However, ETS becomes very much different from side-stream smoke as it ages and undergoes complex chemical and physical transformations by interacting with oxygen, light, and many other environmental components (2-5).

Nominally, then, ETS and mainstream smoke may share some components, but their chemical and physical properties are substantially different, as the National Academy of Sciences has recognized (4). Moreover, the presence of most ETS components can only be postulated because most are beyond material detection, and only around three dozen are analytically traceable to ETS under real life conditions (1-5).

In view of this situation, there is no scientific justification for the regulation of ETS in workplaces on the basis of its comparison with main-stream smoke. Even under the unwarranted assumption that side-stream smoke remains unaltered as it becomes diluted and aged ETS, concentrations of postulated ETS components would be several orders of magnitude below the corresponding PEL or TWA levels permitted in workplaces by the U.S. Occupational Safety and Health Administration (8). Indeed, the EPA report itself presents data suggesting that in the course of an entire year the average nonsmoker may inhale less "tar" than the active smoking of one single cigarette (1,8).

Thus, the vanishing nature of ETS precludes postulating its similarity to main-stream smoke beyond vague conjectures. The Agency's Science Advisory Board itself told EPA's staff that the alleged equivalency of ETS and cigarette smoke could not be sustained (9). Still, EPA's contrived identification of ETS and main-stream smoke was felt necessary to bolster the weak and inconclusive results of ETS epidemiologic studies.

EPA'S CLAIM THAT ETS IS A LUNG CANCER RISK STEMS FROM THE SELECTIVE AND IMPROPER USE OF EPIDEMIOLOGIC DATA. EPIDEMIOLOGIC REPORTS OF WORKPLACE ETS EXPOSURE DO NOT SUPPORT CLAIMS OF INCREASED LUNG CANCER RISK.

Three categories of exposure can be identified in epidemiologic studies of ETS and lung cancer. One group considers people exposed to ETS at work, and shows no overall increase of lung cancer risk. MOSHAB should be especially interested in this absence of overall risk as it comes from 13 published studies on lung cancer and workplace exposure to ETS, of which 7 were conducted in the U.S (Figure 1). Several other studies have reported no increase of risk for workplace exposures without providing specific data (*).

A second exposure category in epidemiologic studies addresses people exposed to ETS at home since childhood, and the combined assessment from these studies shows no overall increase of lung cancer risk (Figure 2). This result comes despite general claims by EPA and other agencies that children are an especially susceptible lot. Adopting an unjustifiable selectivity, the EPA decided not to consider data from these and the negative workplace studies in its risk assessment exercise.

(*) MOSHAB should also be aware of certain inconsistent published reviews of workplace studies that have not specifically addressed ETS exposures. For instance, a recent review by Siegel reports that " (s)moking rates in cooks are 30% higher than in the general male population"(10). The same reviewer also reports that, after controlling for active smoking, cooks as a group do not show an increased risk of lung cancer, suggesting that ETS is not a risk factor. With the possible exception of nicotine, known ETS components are also found in much higher concentrations in the smokes, fumes, and gases to which cooks are occupationally and continuously exposed. Siegel then proceeds to make the implausible – and in fact contradictory – inference that ETS might be responsible for the slight increase of lung cancer risk reported for restaurant workers in general, a group that by his own admission should be exposed to less ETS and known environmental hazards than cooks themselves. Obviously, if the slight increase of risk for restaurant workers is real, it may be due to factors other than ETS.

A third category of exposure in epidemiologic studies involves some 30 studies from all over the world, which address nonsmoking wives reportedly exposed to the ETS generated by their smoking husbands at home. Of these studies, some reported a small increase in risk, some no risk change at all, and some actually reported a decrease of risk (Figure 3). The EPA based its risk assessment on 11 US spousal exposure studies, none of which reports an overall statistically significant increase of risk at the 95% confidence level. The results of these studies are compatible with either an increase or a decrease of risk (1).

I would like to make clear that this not just my opinion about epidemiologic data. It is also the conclusion reached by the International Agency for Research on Cancer – an agency of the World Health Organization – which stated in 1986:

"... [S]ubstantial difficulties in [the] determination of passive exposure to tobacco smoke and to other possible risk factors [exist]. The resulting errors could arguably have artificially depressed or raised estimated risks, and, as a consequence, each is compatible either with an increase or with an absence of risk." (11)

EPA's further arbitrary decision was to ignore the two latest completed studies on ETS, funded by the National Cancer Institute and reporting data that do not agree with the Agency's conclusions (12,13).

The inconsistency of epidemiologic studies derives from the many problems they share. It is difficult if not impossible to define with any credibility the lifetime exposure to ETS of any person, especially deceased ones. More important, studies show that on the average 4% to 5% of self reported nonsmokers misrepresent their status and are in fact smokers. That proportion may be considerably greater when the subjects interviewed are lung cancer patients, as in the case of ETS studies (14,15). To reach its preconceived

conclusions, EPA used an arbitrarily low misclassification adjustment, when a realistic one – even one below average reported rates -- would have voided any reports of increased risk (1,14).

There is more. Despite much epidemiologic evidence to the contrary, EPA largely discounted manifest confounders of lung cancer risk, such as differences in nutrition, disease experiences, physical activity, socioeconomic status, occupation, and others. The scientific literature reports over two dozen independent risk factors for lung cancer, and these confounders are more than capable of accounting for the small risk attributed to ETS by the Agency (8,14). One cannot look at ETS alone, as the agency has apparently done. It is as if police came upon a case involving several people, but arrested only one because he "looked" suspicious.

Also, EPA subverted accepted statistical standards, and doubled from 5% to 10% the margin of error it usually accepts in determining statistical significance (1). The arbitrariness of this gambit is apparent, considering that the Agency would refuse to consider regulatory petitions based on less than the 5% standard.

It should be made clear that the Agency did not conduct studies of its own: it simply reviewed studies performed by others. Despite results compatible with increased or decreased risk, the Agency arbitrarily decided to consider only increased risk. Indeed, the EPA report states that its conclusions were "...based on the a priori hypothesis...that a positive association exists between exposure to ETS and lung cancer."(1, page 5-2). Actually, the Agency's prejudice against ETS was made public in official documents as early as 1989 (16).

Thus, EPA's claim that ETS causes 3060 lung cancers a year is based on unwarranted assumptions, selective use of data, artful procedural manipulations, and the contrived illusion of mathematical precision (1). The EPA Science Advisory Board itself advised

the Agency against producing numerical estimates (17). Dr. Erich Bretthauer, then Associate Administrator for R&D at EPA had to admit in official correspondence that the excess risk of lung cancer could also be virtually zero (18). In fact, there was dissent within EPA itself, and prominent internal EPA reviewers documented their criticism of the Agency's report (19)

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The EPA report could not find reason to address ETS exposure as a possible cardiovascular risk. Other reviewers, however, have contrived to make such claims. Steenland, the most quoted of these reviewers, estimated at 1.3 the average excess risk from a pool of nine epidemiologic studies of cardiovascular diseases and ETS (20). Several individual studies, however, give reduced risk reports (14,20).

To understand the meaning of these pooled values, it is important to recall that the apparent risk of cardiovascular diseases for active smokers is only 1.7. And even for active smokers there are solid reports of negative cardiovascular risks in the Framingham female sample (21), among British doctors (22), and in a large Swedish study (23).

Steenland sensed the paradox and rushed to assert that "arguments inferring ETS health effects based on known health effects of mainstream smoke...are not appropriate", in contradiction to official EPA assertions (20). Indeed, such inference would be disturbing because the apparent reported risk in active smokers is only slightly higher than the

reported risk from ETS studies, despite vastly smaller ETS doses. The closeness of the reported risk values indicates either the unlikely hypothesis of extreme differences in the specific biologic potencies of ETS and main-stream smoke, or the more plausible likelihood of interferences from confounding risk factors other than ETS. Steenland writes:

"Due to the relatively slight increased risk of heart disease for passive smokers and the many factors known to affect heart disease, the possibility of uncontrolled confounding as a cause for the increased risk cannot be ruled out." (20).

In fact, the role of confounders is certain, given that over 250 risk factors are reported in the literature and only a few of the nine studies of ETS and cardiovascular diseases have controlled at most for two or three of these factors (8,20). Nonetheless, setting aside all troubling considerations raised in his own paper, Steenland calculates ischemic heart disease deaths attributed to ETS with digital accuracy ranging from 74 to 18,390 units (20). Without explanation or supporting data, he assumes that the epidemiologic evidence is "reasonably accurate," that misclassifications and confounders are not "likely accounts for the observed risks," and that epidemiologic data are valid because "multiple studies are now consistent and reasonably well designed."

Objectively, these assumptions and conclusions are incompatible with the data. The consistency of some -- not all – available studies more likely reflects consistent simplistic designs, consistent disregard of confounders, and consistent likelihood of bias. In fact, the objections reviewed above in regard to lung cancer studies are equally valid objections to the execution and interpretation of epidemiologic studies of ETS and cardiovascular risk.

THE SURGEON GENERAL AND OTHERS REPORT NO STATISTICALLY SIGNIFICANT ASSOCIATION OF ETS AND RESPIRATORY DISEASES IN ADULTS

No clear association of ETS and respiratory diseases in adults has been established, despite many studies. The US Surgeon General Report on ETS (5) stated:

".. a previously healthy individual would not develop chronic lung disease solely on the basis of [ETS exposure in adult life]". Regarding airflow obstruction, the same report noted "quite small" change in test subjects, and stated: "..it is unlikely that this change in airflow, per se, results in symptoms."

Parallel conclusions were drawn by the National Academy of Sciences (6). Subsequent studies confirmed the situation even for highly susceptible individuals, prompting the following assessment by Samet, a prominent ETS opponent:

"Neither epidemiological nor experimental studies have established the role of ETS in exacerbating asthma in adults..... Other studies have not shown chronic effects of involuntary exposure to tobacco smoke in adult nonsmokers."(24).

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I wish to make it clear that my comments should not be interpreted to imply that ETS entails no risk at all. The essence of my critique is that the epidemiologic data are too uncertain to draw any conclusion that is scientifically justified. This critique applies to individual studies, as well as to other published reviews, such as the one by Steenland claiming an association of ETS and cardiovascular diseases (20), and the one by Siegel, claiming an increased risk for restaurant workers (10).

EPA claims to have used a "weight of evidence" approach. However, the agent in question -- ETS -- has been characterized only indirectly by a proxy that EPA itself later discounts. Many other agents should be considered with even greater reason, but were summarily ignored or dismissed. There are irrefutable alibis of misclassification, and no conclusive proof. By its own admission, EPA made a very selective use of the available studies, emphasizing only those that support its preconceived objectives.

Despite these fatal shortcomings as an objective scientific document -- and although EPA asserts to have no regulatory aims -- the Agency's ETS report has been invoked uncritically at face value by all levels of federal and local governments in support of strict regulations to eliminate ETS in public and work places. Many have attempted to describe ETS as a major issue in indoor air quality, and a prime factor in what has come to be known as the "sick building syndrome".

In reality, published data show that in normal situations nonsmoker reactions to ETS depend more on psychosomatic and culturally determined aversions than on actual irritation (25,26). The National Institute for Occupational Safety and Health estimated that less than 2% of sick building complaints might have had an ETS component (27). Other sources of indoor air deterioration are overwhelmingly more prevalent and important than ETS (27,28).

Such liberties with policy and scientific manipulations are part of a pattern of behavior at EPA. In 1991, an independent blue ribbon panel was convened by William Reilly, the former EPA Administrator, to investigate accusations of "junk science" at the Agency. The panel concluded that the Agency does not have a coherent science agenda and suffers from poor scientific credibility because it has not secured adequate external support from the scientific community at large. More to the point, the panel concluded that EPA's science often seems to have been adjusted to endorse policy, an especially relevant statement when considering the Agency's report on ETS (29).

A regulatory agency does not fulfill the public expectations of due process and fairness, if it operates on the basis of unwarranted assumptions. At the very least, it should obey generally accepted norms of scientific evidence and conduct: norms that are independently justified by their transparent and rational honesty and timeless ethical value.

In the case of ETS, objective scientific data do not support a workplace smoking ban, nor do they show that – if enacted – such a ban would impart any demonstrable benefit to workers.

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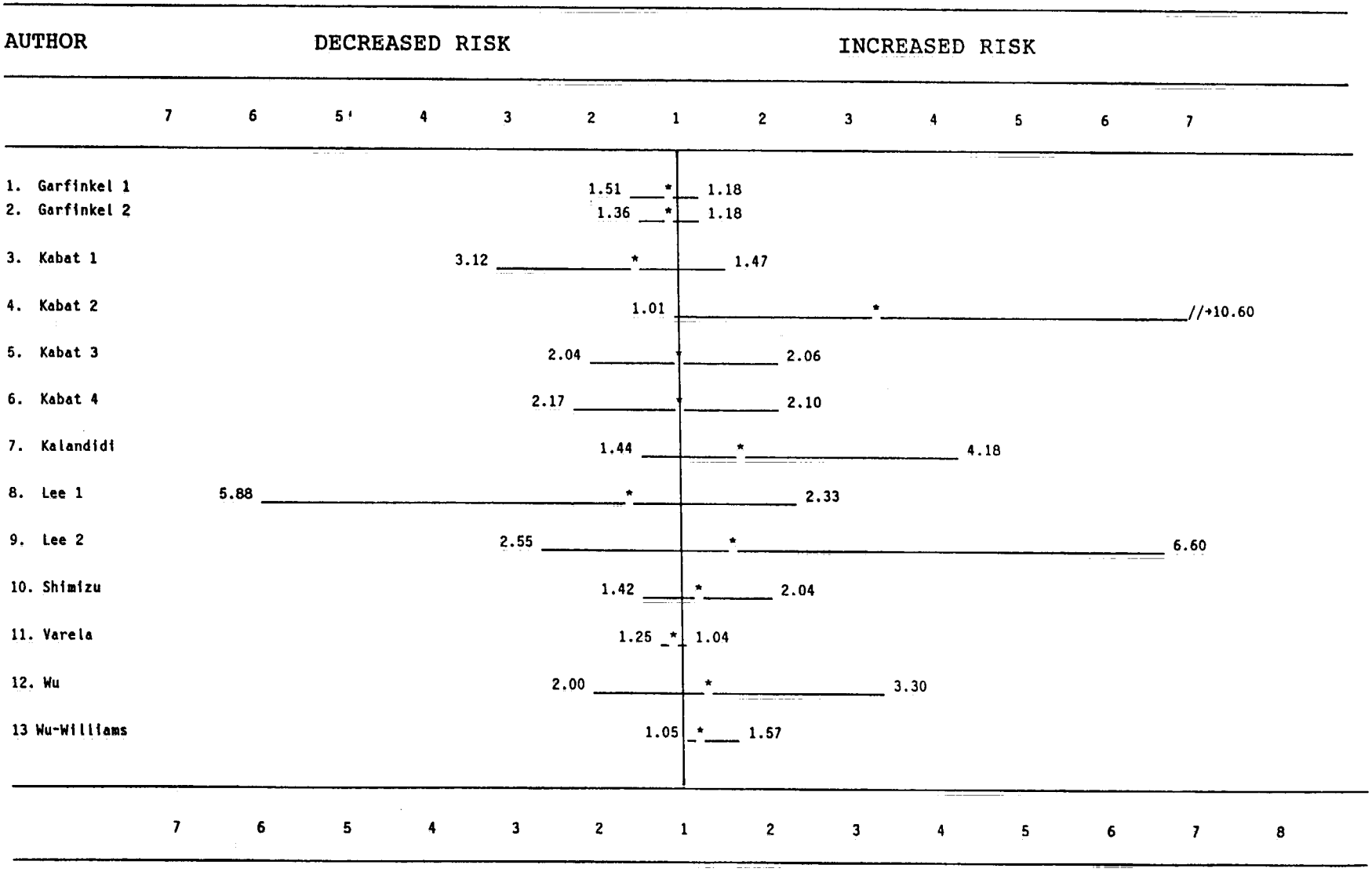
FIGURE 1 *

SYMMETRIC SCALE DISPLAY OF INCREASED AND DECREASED LUNG CANCER RISK
IN NEVER SMOKERS EXPOSED TO ENVIRONMENTAL TOBACCO SMOKE AT WORK

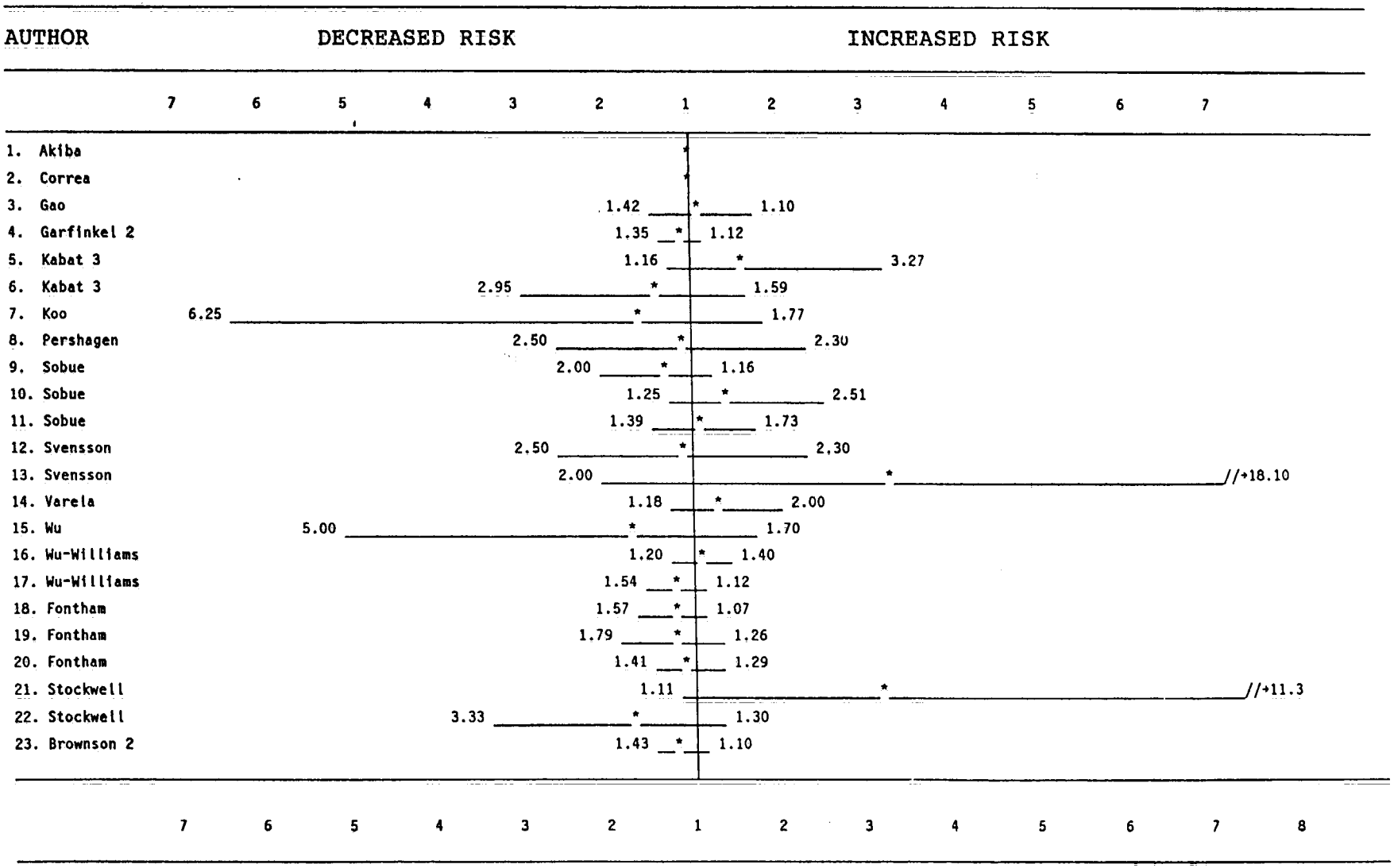


References: 1,14.

FIGURE 2 *

INCREASED AND DECREASED RISK OF LUNG CANCER

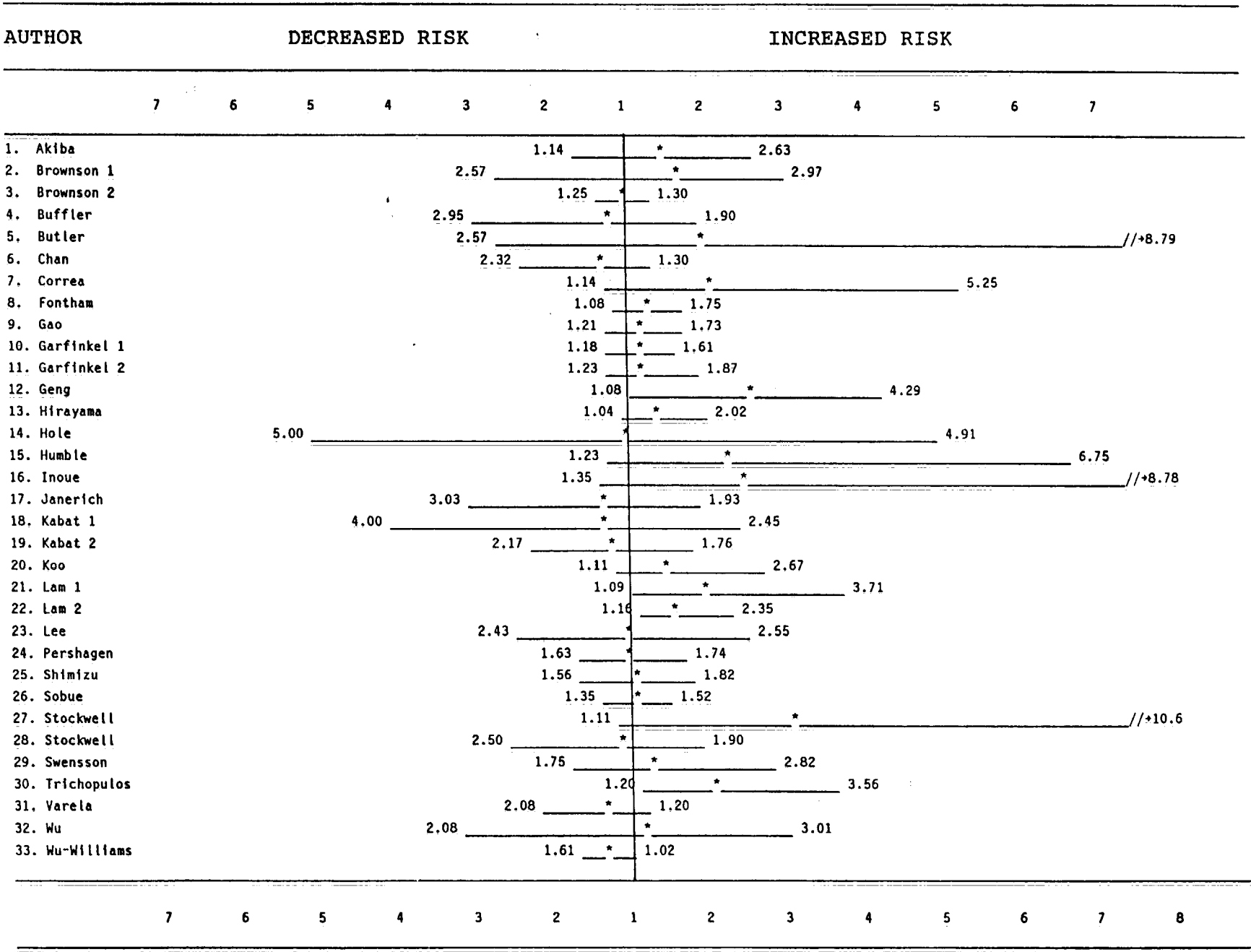
IN NEVER SMOKERS EXPOSED TO ENVIRONMENTAL TOBACCO SMOKE DURING CHILDHOOD



References: 1,12,13,14

FIGURE 3 *

INCREASED AND DECREASED LUNG CANCER RISK IN NEVER SMOKING FEMALES MARRIED TO SMOKERS



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